

"HELP COMMANDS" at an arrow prompt (=>).

=> file registry
COST IN U.S. DOLLARS

SINCE FILE ENTRY	TOTAL SESSION
0.21	0.21

FULL ESTIMATED COST

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STRUCTURE FILE UPDATES: 6 JUL 2006 HIGHEST RN 890869-30-4
DICTIONARY FILE UPDATES: 6 JUL 2006 HIGHEST RN 890869-30-4

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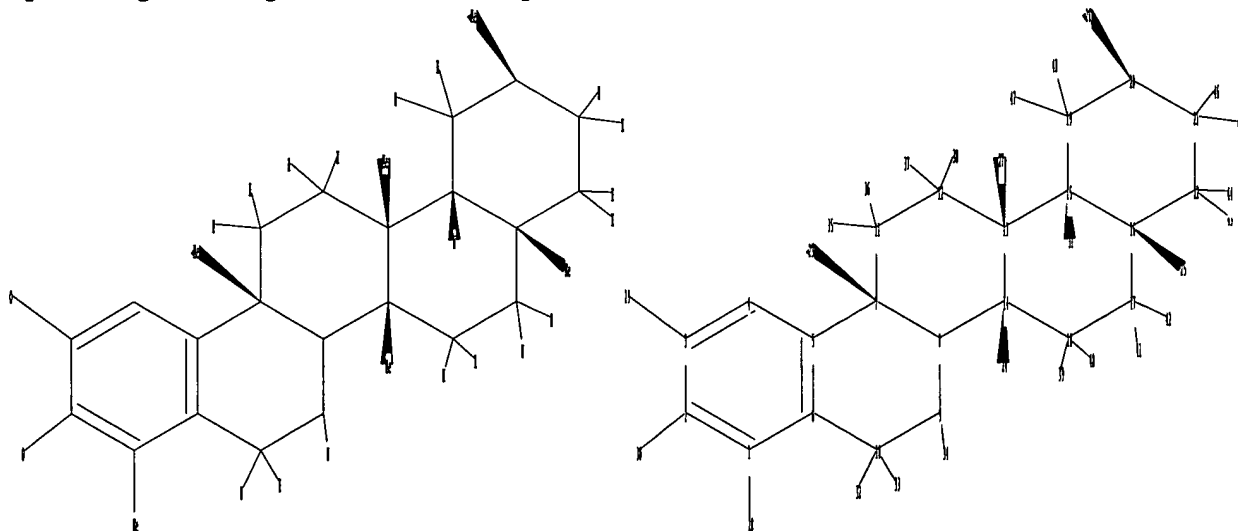
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=>
Uploading C:\Program Files\Stnexp\Queries\10773903core2.str



chain nodes :
23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43
44 45 46 47 48
ring nodes :
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22
chain bonds :
1-28 2-30 3-29 7-23 9-34 10-32 10-33 11-35 11-36 12-37 12-38 13-27
14-24 15-31 16-25 17-41 17-42 18-39 18-40 19-47 19-48 20-26 21-45 21-46
22-43 22-44

```

ring bonds :
1-2 1-6 2-3 3-4 4-5 5-6 5-7 6-10 7-8 7-11 8-9 8-14 9-10 11-12 12-13
13-14 13-15 14-18 15-16 15-19 16-17 16-22 17-18 19-20 20-21 21-22
exact/norm bonds :
2-30 3-29 5-7 6-10 7-8 7-11 8-9 8-14 9-10 11-12 12-13 13-14 13-15
14-18 15-16 15-19 16-17 16-22 17-18 19-20 20-21 21-22
exact bonds :
1-28 7-23 9-34 10-32 10-33 11-35 11-36 12-37 12-38 13-27 14-24 15-31
16-25 17-41 17-42 18-39 18-40 19-47 19-48 20-26 21-45 21-46 22-43 22-44
normalized bonds :
1-2 1-6 2-3 3-4 4-5 5-6

```

Match level :

```

1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:Atom 8:Atom 9:Atom 10:Atom
11:Atom 12:Atom 13:Atom 14:Atom 15:Atom 16:Atom 17:Atom 18:Atom 19:Atom
20:Atom 21:Atom 22:Atom 23:CLASS 24:CLASS 25:CLASS 26:CLASS 27:CLASS
28:CLASS 29:CLASS 30:CLASS 31:CLASS 32:CLASS 33:CLASS 34:CLASS 35:CLASS
36:CLASS 37:CLASS 38:CLASS 39:CLASS 40:CLASS 41:CLASS 42:CLASS 43:CLASS
44:CLASS 45:CLASS 46:CLASS 47:CLASS 48:CLASS

```

Stereo Bonds:

```

23-7 (Single Wedge).
24-14 (Single Wedge).
25-16 (Single Wedge).
26-20 (Single Wedge).
27-13 (Single Hash).
31-15 (Single Wedge).

```

Stereo Chiral Centers:

```

7      (Parity=Even)
13     (Parity=Odd)
14     (Parity=Odd)
15     (Parity=Even)
16     (Parity=Even)
20     (Parity=Odd)

```

Stereo RSS Sets:

Type=Relative (Default). 6 Nodes= 7 13 14 15 16 20

L1 STRUCTURE UPLOADED

=> s L1

SAMPLE SEARCH INITIATED 15:52:44 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED - 816 TO ITERATE

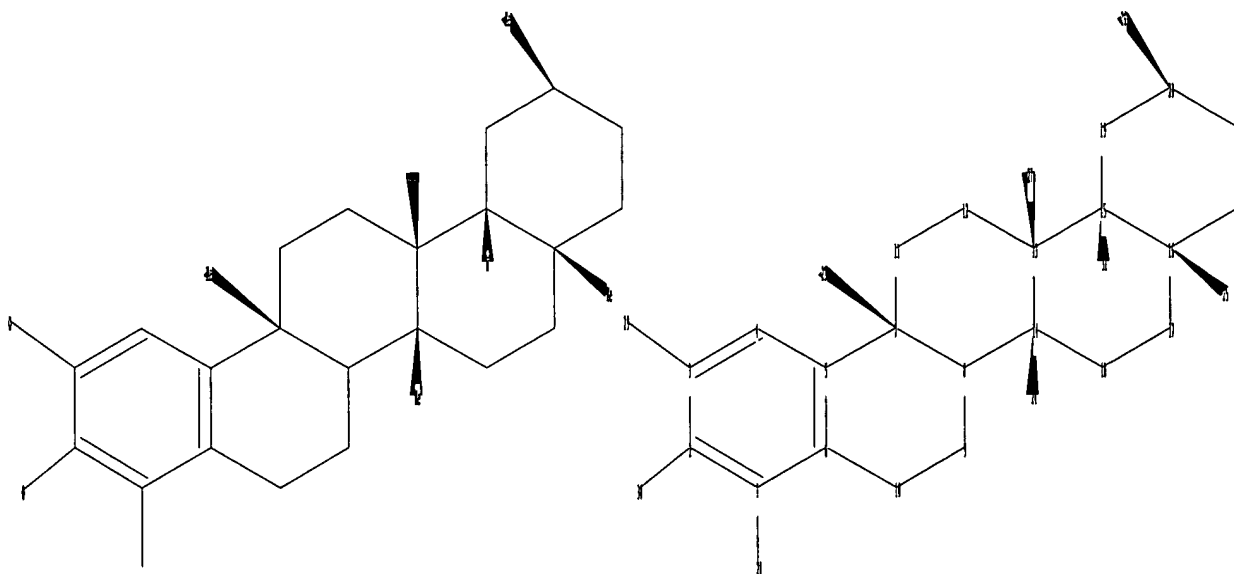
100.0% PROCESSED 816 ITERATIONS 0 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**
BATCH **COMPLETE**
PROJECTED ITERATIONS: 14607 TO 18033
PROJECTED ANSWERS: 0 TO 0

L2 0 SEA SSS SAM L1

=>

Uploading C:\Program Files\Stnexp\Queries\10773903core.str



```

chain nodes :
23 24 25 26 27 28 29 30 31
ring nodes :
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22
chain bonds :
1-28 2-30 3-29 7-23 13-27 14-24 15-31 16-25 20-26
ring bonds :
1-2 1-6 2-3 3-4 4-5 5-6 5-7 6-10 7-8 7-11 8-9 8-14 9-10 11-12 12-13
13-14 13-15 14-18 15-16 15-19 16-17 16-22 17-18 19-20 20-21 21-22
exact/norm bonds :
2-30 3-29 5-7 6-10 7-8 7-11 8-9 8-14 9-10 11-12 12-13 13-14 13-15
14-18 15-16 15-19 16-17 16-22 17-18 19-20 20-21 21-22
exact bonds :
1-28 7-23 13-27 14-24 15-31 16-25 20-26
normalized bonds :
1-2 1-6 2-3 3-4 4-5 5-6

```

```

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:Atom 8:Atom 9:Atom 10:Atom
11:Atom 12:Atom 13:Atom 14:Atom 15:Atom 16:Atom 17:Atom 18:Atom 19:Atom
20:Atom 21:Atom 22:Atom 23:CLASS 24:CLASS 25:CLASS 26:CLASS 27:CLASS
28:CLASS 29:CLASS 30:CLASS 31:CLASS

```

Stereo Bonds:

```

23-7 (Single Wedge).
24-14 (Single Wedge).
25-16 (Single Wedge).
26-20 (Single Wedge).
27-13 (Single Hash).
31-15 (Single Wedge).

```

Stereo Chiral Centers:

```

7      (Parity=Even)
13     (Parity=Odd)
14     (Parity=Odd)
15     (Parity=Even)
16     (Parity=Even)

```

20 (Parity=Odd)

Stereo RSS Sets:

Type=Relative (Default). 6 Nodes= 7 13 14 15 16 20

L3 STRUCTURE UPLOADED

=> s L3

SAMPLE SEARCH INITIATED 15:53:11 FILE 'REGISTRY'

SAMPLE SCREEN SEARCH COMPLETED - 816 TO ITERATE

100.0% PROCESSED 816 ITERATIONS

0 ANSWERS

SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**

BATCH **COMPLETE**

PROJECTED ITERATIONS: 14607 TO 18033

PROJECTED ANSWERS: 0 TO 0

L4 0 SEA SSS SAM L3

=> file bioscience patents

'BIOSCIENSE' IS NOT A VALID FILE NAME

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ENTER A FILE NAME OR (IGNORE):bioscience

FILE 'ENCOMPPAT2' ACCESS NOT AUTHORIZED

FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED

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SINCE FILE

TOTAL

ENTRY

SESSION

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0.88

1.09

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COST IN U.S. DOLLARS

	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	121.89	122.98

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 15:53:47 ON 07 JUL 2006

92 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
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=> s heat(w)shock(w)protein

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68	FILE ADISINSIGHT
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21128	FILE BIOSIS
967	FILE BIOTECHABS
967	FILE BIOTECHDS
9367	FILE BIOTECHNO
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14692	FILE CAPLUS
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78	FILE PROUSDDR
1	FILE RDISCLOSURE

```

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45     FILE WATER
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68 FILES SEARCHED...
10     FILE CASREACT
251    FILE DPCI
1      FILE ENCOMPPAT
682    FILE EPFULL
7      FILE FRANCEPAT
24     FILE FRFULL
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43     FILE IMSPATENTS
605    FILE INPADOC
98     FILE JAPIO
17     FILE KOREAPAT
24     FILE PAPERCHEM2
5      FILE PATDPA
69     FILE PATDPAFULL
86 FILES SEARCHED...
2972   FILE PCTFULL
1      FILE PIRA
1      FILE RAPRA
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2      FILE TULSA
1      FILE TULSA2

```

75 FILES HAVE ONE OR MORE ANSWERS, 92 FILES SEARCHED IN STNINDEX

L5 QUE HEAT(W) SHOCK(W) PROTEIN

```

=> file biosis embase medline scisearch biotechno caplus
COST IN U.S. DOLLARS                               SINCE FILE      TOTAL
                                                    ENTRY      SESSION
FULL ESTIMATED COST                               2.44        125.42

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```

=> s heat(w)shock(w)protein
L6      92930 HEAT(W) SHOCK(W) PROTEIN

```

```

=> s L6 and inflamm?
L7      5787 L6 AND INFLAMM?

```

=> s L7 not py>2003
L8 4016 L7 NOT PY>2003

=> s L7 not py>2002
L9 3307 L7 NOT PY>2002

=> s L9 and celastrol
L10 0 L9 AND CELASTROL

=> s L9 and dihydrocelastrol
L11 0 L9 AND DIHYDROCELASTROL

=> s L6 and (cancer or neoplas?)
L12 9519 L6 AND (CANCER OR NEOPLAS?)

=> s L6 and (neurodegener? or Alzheimer or parkinson)
L13 2020 L6 AND (NEURODEGENER? OR ALZHEIMER OR PARKINSON)

=> s L7 and L12 and L13
L14 35 L7 AND L12 AND L13

=> s L14 not py>2002
L15 10 L14 NOT PY>2002

=> dup rem L15
PROCESSING COMPLETED FOR L15
L16 7 DUP REM L15 (3 DUPLICATES REMOVED)

=> d L16 1-7 ti

L16 ANSWER 1 OF 7 MEDLINE on STN
TI Prostaglandin E synthase.

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TI The biochemistry and medical significance of the flavonoids.

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TI HSP105 IS UP - REGULATED BY NEUROTOXIC PROSTAGLANDINS D2 AND J2 IN MOUSE AND HUMAN NEUROBLASTOMA CELLS.

L16 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Heat-shock proteins: New keys to the development of cytoprotective therapies

L16 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Clinical application of heat shock proteins

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TI Stress-inducible responses and heat shock protein: New pharmacologic targets for cytoprotection.

L16 ANSWER 7 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 1
TI Immunohistochemical study of the expression of human groEL-stress protein in human nervous tissue.

=> d L16 1-7 ti abs bib

L16 ANSWER 1 OF 7 MEDLINE on STN
TI Prostaglandin E synthase.
AB Prostaglandin E synthase (PGES), which converts cyclooxygenase (COX)-derived prostaglandin (PG)H2 to PGE2, occurs in multiple forms with

distinct enzymatic properties, modes of expression, cellular and subcellular localizations and intracellular functions. Cytosolic PGES (cPGES) is a cytosolic protein that is constitutively expressed in a wide variety of cells and tissues and is associated with heat shock protein 90 (Hsp90). Membrane-associated PGES (mPGES), the expression of which is stimulus-inducible and is downregulated by anti-inflammatory glucocorticoids, is a perinuclear protein belonging to the microsomal glutathione S-transferase (GST) family. These two PGESs display distinct functional coupling with upstream COXs in cells; cPGES is predominantly coupled with the constitutive COX-1, whereas mPGES is preferentially linked with the inducible COX-2. Several cytosolic GSTs also have the capacity to convert PGH2 to PGE2 in vitro. Accumulating evidence has suggested that mPGES participates in various pathophysiological states in which COX-2 is involved, implying that mPGES represents a potential novel target for drug development.

AN 2002672251 MEDLINE
 DN PubMed ID: 12432931
 TI Prostaglandin E synthase.
 AU Murakami Makoto; Nakatani Yoshihito; Tanioka Toshihiro; Kudo Ichiro
 CS Department of Health Chemistry, School of Pharmaceutical Sciences, Showa University, Japan.. mako@pharm.showa-u.ac.jp
 SO Prostaglandins & other lipid mediators, (2002 Aug) Vol. 68-69, pp. 383-99. Ref: 83
 Journal code: 9808648. ISSN: 1098-8823.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 LA English
 FS Priority Journals
 EM 200307
 ED Entered STN: 16 Nov 2002
 Last Updated on STN: 11 Jul 2003
 Entered Medline: 10 Jul 2003

L16 ANSWER 2 OF 7 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

TI The biochemistry and medical significance of the flavonoids.
 AB Flavonoids are plant pigments that are synthesised from phenylalanine, generally display marvelous colors known from flower petals, mostly emit brilliant fluorescence when they are excited by UV light, and are ubiquitous to green plant cells. The flavonoids are used by botanists for taxonomical classification. They regulate plant growth by inhibition of the exocytosis of the auxin indolyl acetic acid, as well as by induction of gene expression, and they influence other biological cells in numerous ways. Flavonoids inhibit or kill many bacterial strains, inhibit important viral enzymes, such as reverse transcriptase and protease, and destroy some pathogenic protozoans. Yet, their toxicity to animal cells is low. Flavonoids are major functional components of many herbal and insect preparations for medical use, e.g., propolis (bee's glue) and honey, which have been used since ancient times. The daily intake of flavonoids with normal food, especially fruit and vegetables, is 1-2 g. Modern authorised physicians are increasing their use of pure flavonoids to treat many important common diseases, due to their proven ability to inhibit specific enzymes, to simulate some hormones and neurotransmitters, and to scavenge free radicals. .COPYRGT. 2002 Elsevier Science Inc. All rights reserved.

AN 2002423363 EMBASE
 TI The biochemistry and medical significance of the flavonoids.
 AU Havsteen B.H.
 CS B.H. Havsteen, Abildgaardsvej 49, DK-2830 Virum, Denmark. benthavs@worldonline.dk
 SO Pharmacology and Therapeutics, (2002) Vol. 96, No. 2-3, pp. 67-202. . Refs: 1333
 ISSN: 0163-7258 CODEN: PHTHDT

PUI S 0163-7258(02)00298-X
CY United States
DT Journal; Article
FS 030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
ED Entered STN: 12 Dec 2002
Last Updated on STN: 12 Dec 2002

L16 ANSWER 3 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI HSP105 IS UP - REGULATED BY NEUROTOXIC PROSTAGLANDINS D2 AND J2 IN MOUSE
AND HUMAN NEUROBLASTOMA CELLS.

AB In many neurodegenerative disorders, aggregates of ubiquitinated proteins accumulate in neuronal inclusions. The mechanisms forming such abnormal aggregates are unclear and their role in disease progression has yet to be elucidated. We previously showed that some prostaglandins (PGs) are potent neurotoxins in mouse HT4 and human SK-N-SH neuroblastoma cells. PGA1, D2 and J2, but not E2, promoted a dose-dependent decrease in neuronal cell viability and an increase in ubiquitinated protein aggregates. We attempted to identify molecules that may promote cell survival in response to the neurotoxic PGs. Heat shock proteins (HSPs) were likely candidates, since they are known to have neuroprotective functions by promoting protein folding and preventing their aggregation. HSP105 is one of the most abundant proteins in the brain, but its actions in neurodegenerative disorders are not well understood. Presently, we demonstrate that, in mouse HT4 and human SK-N-SH neuroblastoma cells, the protein levels of HSP105 are dramatically up-regulated in a concentration-dependent fashion by PGD2 and J2, the most toxic of the PGs tested in our studies. These findings suggest that HSP105 may have a neuroprotective role under pro-inflammatory conditions that cause an increase in the levels of ubiquitinated proteins. Further elucidation of the roles played by HSP105 in neuroprotection and identification of its putative protein partners may uncover new targets for therapeutic intervention in neuronal diseases as well as diagnostic markers for individuals at risk for these disorders.

AN 2003:326978 BIOSIS
DN PREV200300326978
TI HSP105 IS UP - REGULATED BY NEUROTOXIC PROSTAGLANDINS D2 AND J2 IN MOUSE
AND HUMAN NEUROBLASTOMA CELLS.
AU Pierre, S. [Reprint Author]; Hunter, L. [Reprint Author]; Johnston, J. M.;
Tezapsidis, N.; Figueiredo-Pereira, M. E. [Reprint Author]
CS Biol.Sc., Hunter College, NY, NY, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002)
Vol. 2002, pp. Abstract No. 785.18. <http://sfn.scholarone.com>. cd-rom.
Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience.
Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 16 Jul 2003
Last Updated on STN: 16 Jul 2003

L16 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Heat-shock proteins: New keys to the development of cytoprotective
therapies

AB A review. All cells, from bacterial to human, have a common, intricate response to stress that protects them from injury. Heat-shock proteins (Hsps), also known as stress proteins and mol. chaperones, play a central role in protecting cellular homeostatic processes from environmental and physiol. insult by preserving the structure of normal proteins and repairing or removing damaged ones. An understanding of the interplay between Hsps and cell stress tolerance will provide new tools for

treatment and drug design that maximize the preservation or restoration of health. For example, the increased vulnerability of tissues to injury in some conditions, such as ageing, diabetes mellitus, and menopause, or with the use of certain drugs, such as some antihypertensive medications, is associated with an impaired Hsp response. Addnl., diseases that are associated with tissue oxidation, free radical formation, disorders of protein folding, or inflammation, may be improved therapeutically by elevated expression of Hsps. The accumulation of Hsps, whether induced physiol., pharmacol., genetically, or by direct administration of the proteins, is known to protect the organism from a great variety of pathol. conditions, including myocardial infarction, stroke, sepsis, viral infection, trauma, neurodegenerative diseases, retinal damage, congestive heart failure, arthritis, sunburn, colitis, gastric ulcer, diabetic complications, and transplanted organ failure. Conversely, lowering Hsps in cancer tissues can amplify the effectiveness of chemo- or radiotherapy. Treatments and agents that induce Hsps include hyperthermia, heavy metals (zinc and tin), salicylates, dexamethasone, cocaine, nicotine, alc., α -adrenergic agonists, PPAR- γ agonists, Bimoclomol, Geldanamycin, geranylgeranylacetone, and cyclopentenone prostanoids. Compds. that suppress Hsps include quercetin (a bioflavonoid), 15-deoxyspergualin (an immunosuppressive agent), and retinoic acid. Researchers who are cognizant of the Hsp-related effects of these and other agents will be able to use them to develop new therapeutic paradigms.

AN 2001:383675 CAPLUS
 DN 136:111904
 TI Heat-shock proteins: New keys to the development of cytoprotective therapies
 AU Tytell, Michael; Hooper, Philip L.
 CS Department of Neurobiology and Anatomy, Wake Forest University School of Medicine, Winston-Salem, NC, 27157, USA
 SO Emerging Therapeutic Targets (2001), 5(2), 267-287
 CODEN: ETTF7; ISSN: 1460-0412
 PB Ashley Publications Ltd.
 DT Journal; General Review
 LA English
 RE.CNT 201 THERE ARE 201 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
 TI Clinical application of heat shock proteins
 AB A review with 50 refs. Heat shock proteins (Hsps) comprise a family of ubiquitous and evolutionary conserved proteins playing a fundamental biol. role both under stress conditions and during normal growth, development and differentiation. During the last decade, the knowledge about their expression and cellular functions has rapidly accumulated providing the basis for the increasing clin. application of these proteins. The expression of Hsps in different cells and tissues is associated with the etiol. and/or progress of a number of diseases such as cerebrovascular, cardiovascular, neurodegenerative, autoimmune and malignant diseases, various infections and inflammatory reactions. The present review summarizes the possibilities of clin. application of Hsps as prognostic, diagnostic and therapeutic tools as well as stress monitoring parameters in toxicol. and public health.
 AN 2000:44667 CAPLUS
 DN 132:206077
 TI Clinical application of heat shock proteins
 AU Matic, Gordana
 CS Department of Biochemistry, Institute of Biological Research, Belgrade, 11060, Yugoslavia
 SO Jugoslovenska Medicinska Biohemija (1999), 18(4), 133-139
 CODEN: JMBIFF; ISSN: 0354-3447
 PB Drustvo Medicinskih Biohemicara Jugoslavije
 DT Journal; General Review
 LA English

RE.CNT 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 6 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Stress-inducible responses and heat shock
protein: New pharmacologic targets for cytoprotection.
AB Molecular chaperones protect proteins against environmental and
physiologic stress and from the deleterious consequences of an imbalance
in protein homeostasis. Many of these stresses, if prolonged, result in
defective development and pathologies associated with a diverse array of
diseases due to tissue injury and repair including stroke, myocardial
reperfusion damage, ischemia, cancer, amyloidosis, and other
neurodegenerative diseases. We discuss the molecular nature of
the stress signals, the mechanisms that underlie activation of the heat
shock response, the role of heat shock proteins as cytoprotective
molecules, and strategies for pharmacologically active molecules as
regulators of the heat shock response.
AN 1998:473294 BIOSIS
DN PREV199800473294
TI Stress-inducible responses and heat shock
protein: New pharmacologic targets for cytoprotection.
AU Morimoto, Richard I. [Reprint author]; Santoro, M. Gabriella
CS Dep. Biochemistry Molecular Biology Cell Biology, Rice Inst. Biomedical
Res., Northwestern Univ., Evanston, IL 60208, USA
SO Nature Biotechnology, (Sept., 1998) Vol. 16, No. 9, pp. 833-838. print.
ISSN: 1087-0156.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 5 Nov 1998
Last Updated on STN: 5 Nov 1998

L16 ANSWER 7 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 1
TI Immunohistochemical study of the expression of human groEL-stress protein
in human nervous tissue.
AB Monoclonal antibody (ML-30) directed against 65 kDa stress protein of
mycobacteria, is shown to identify human cellular protein homologous with
the groEL heat shock protein in many
prokaryotes. Immunohistochemical survey of nervous tissue, both central
and peripheral, from patients dying of various inflammatory,
degenerative and neoplastic conditions and from experimental
animals, using this antibody showed punctate granular staining of the
cells to a variable degree. The astrocytes showed strong immunolabelling.
The normal neurons and oligodendroglia stained variably, while abnormal
neurons were darkly labelled. Ependymal cells showed apical granular
positivity. The ubiquitinated inclusion bodies in amyotrophic lateral
sclerosis, Alzheimer's disease and Parkinson's disease
were not recognised by the ML-30 antibody. In diseased and stressed
nervous tissue from experimental animals, the expression of the ML-30
recognisable stress protein was variable. The epitope recognised by ML-30
was found stable in postmortem tissues collected up to 36 h after death
and processed for paraffin sectioning, after fixation in formalin for many
years. Enhanced expression of the human groEL stress protein homologue in
mammalian nervous tissue following various forms of stress may play a role
in modulating the extent of tissue damage by autoimmune mechanism because
of its high immunogenic mature and constitutive presence in the cells.
AN 1996:191661 BIOSIS
DN PREV199698747790
TI Immunohistochemical study of the expression of human groEL-stress protein
in human nervous tissue.
AU Khanna, Neelam; Shankar, S. K. [Reprint author]; Chandramuki, A.;
Jagannath, C.
CS Natl. Inst. Mental Health Neurosci., Bangalore 560029, India
SO Indian Journal of Medical Research, (1996) Vol. 103, No. FEB., pp.

103-111.
DT Article
LA English
ED Entered STN: 2 May 1996
Last Updated on STN: 2 May 1996

=> d his

(FILE 'HOME' ENTERED AT 15:52:12 ON 07 JUL 2006)

FILE 'REGISTRY' ENTERED AT 15:52:25 ON 07 JUL 2006

L1 STRUCTURE UPLOADED
L2 0 S L1
L3 STRUCTURE UPLOADED
L4 0 S L3

FILE 'ADISCTI, CAOLD, CAPLUS, CASREACT, CROPU, DGENE, DPCI, ENCOMPPAT, EPFULL, FRANCEPAT, FRFULL, FSTA, GBFULL, IFIPAT, IMSPATENTS, INPADOC, JAPIO, KOREAPAT, LITALERT, NTIS, PAPERCHEM2, PATDD, PATDPA, PATDPAFULL, PATDPASPC, PCTFULL, PCTGEN, PIRA, PROUSDDR, ...' ENTERED AT 15:53:27 ON 07 JUL 2006

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 15:53:47 ON 07 JUL 2006
SEA HEAT (W) SHOCK (W) PROTEIN

228 FILE ADISCTI
68 FILE ADISINSIGHT
13 FILE ADISNEWS
729 FILE AGRICOLA
46 FILE ANABSTR
3 FILE ANTE
36 FILE AQUALINE
266 FILE AQUASCI
444 FILE BIOENG
21128 FILE BIOSIS
967 FILE BIOTECHABS
967 FILE BIOTECHDS
9367 FILE BIOTECHNO
1739 FILE CABA
14692 FILE CAPLUS
106 FILE CEABA-VTB
113 FILE CIN
322 FILE CONFSCI
39 FILE CROPU
1188 FILE DDFU
12990 FILE DGENE
666 FILE DISSABS
1305 FILE DRUGU
208 FILE EMBAL
21268 FILE EMBASE
7049 FILE ESBIODBASE
60 FILE FROSTI
122 FILE FSTA
24414 FILE GENBANK
4 FILE HEALSAFE
732 FILE IFIPAT
107 FILE IMSDRUGNEWS
54 FILE IMSRESEARCH
2832 FILE JICST-EPLUS
30 FILE KOSMET
4213 FILE LIFESCI

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 57 FILE NTIS
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 495 FILE PROMT
 78 FILE PROUSDDR
 1 FILE RDISCLOSURE
 15822 FILE SCISEARCH
 7197 FILE TOXCENTER
 4072 FILE USPATFULL
 357 FILE USPAT2
 19 FILE VETU
 45 FILE WATER
 965 FILE WPIDS
 17 FILE WPIFV
 965 FILE WPINDEX
 10 FILE CASREACT
 251 FILE DPCI
 1 FILE ENCOMPPAT
 682 FILE EPFULL
 7 FILE FRANCEPAT
 24 FILE FRFULL
 18 FILE GBFULL
 43 FILE IMSPATENTS
 605 FILE INPADOC
 98 FILE JAPIO
 17 FILE KOREAPAT
 24 FILE PAPERCHEM2
 5 FILE PATDPA
 69 FILE PATDPAFULL
 2972 FILE PCTFULL
 1 FILE PIRA
 1 FILE RAPRA
 3 FILE RUSSIAPAT
 2 FILE TULSA
 1 FILE TULSA2
 L5 QUE HEAT(W) SHOCK(W) PROTEIN

FILE 'BIOSIS, EMBASE, MEDLINE, SCISEARCH, BIOTECHNO, CAPLUS' ENTERED AT
 15:56:06 ON 07 JUL 2006

L6 92930 S HEAT(W) SHOCK(W) PROTEIN
 L7 5787 S L6 AND INFLAMM?
 L8 4016 S L7 NOT PY>2003
 L9 3307 S L7 NOT PY>2002
 L10 0 S L9 AND CELASTROL
 L11 0 S L9 AND DIHYDROCELASTROL
 L12 9519 S L6 AND (CANCER OR NEOPLAS?)
 L13 2020 S L6 AND (NEURODEGENER? OR ALZHEIMER OR PARKINSON)
 L14 35 S L7 AND L12 AND L13
 L15 10 S L14 NOT PY>2002
 L16 7 DUP REM L15 (3 DUPLICATES REMOVED)

=> logoff

ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF

LOGOFF? (Y)/N/HOLD:y

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
50.75	176.17

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION

CA SUBSCRIBER PRICE

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-1.50

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DICTIONARY FILE UPDATES: 6 JUL 2006 HIGHEST RN 890869-30-4

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predicted properties as well as tags indicating availability of
experimental property data in the original document. For information
on property searching in REGISTRY, refer to:

<http://www.cas.org/ONLINE/UG/regprops.html>

=> s dihydrocelastrol/cn

L1 0 DIHYDROCELASTROL/CN

=> exp dihydrocelastrol/cn

E1 1 DIHYDROCEDRELONE ACETATE/CN
E2 1 DIHYDROCELACINNINE/CN
E3 0 --> DIHYDROCELASTROL/CN
E4 1 DIHYDROCELASTROL DIACETATE/CN
E5 1 DIHYDROCEPHALOMANNINE/CN
E6 1 DIHYDROCEPHALOSTATIN 1/CN
E7 1 DIHYDROCERAMIDASE/CN
E8 1 DIHYDROCERAMIDASE (DICTYOSTELIUM DISCOIDEUM)/CN
E9 1 DIHYDROCERAMIDASE (SACCHAROMYCES CEREVISIAE STRAIN YOR1 GENE
YDC1)/CN
E10 1 DIHYDROCERAMIDE Δ4 DESATURASE/CN
E11 1 DIHYDROCERAMIDE DEACYLASE/CN
E12 1 DIHYDROCERAMIDE DESATURASE/CN

=> s E4

L2 1 "DIHYDROCELASTROL DIACETATE"/CN

=> d L2

L2 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2006 ACS on STN

RN 1262-14-2 REGISTRY

ED Entered STN: 16 Nov 1984

CN 24,25,26-Trinoroleana-1,3,5(10),7-tetraen-29-oic acid,
2,3-bis(acetyloxy)-9,13-dimethyl-, (9β,13α,14β,20α)-
(9CI) (CA INDEX NAME)

OTHER CA INDEX NAMES:

CN 24-Nor-D:A-friedooleana-1,3,5(10),7-tetraen-29-oic acid, 2,3-dihydroxy-,
diacetate (7CI, 8CI)

OTHER NAMES:

CN Dihydrocelastrol diacetate

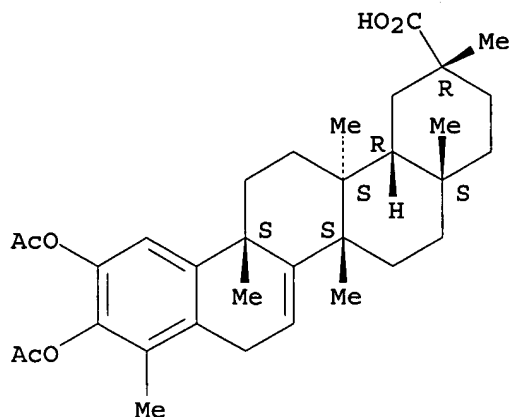
FS STEREOSEARCH

DR 3022-93-3

MF C33 H44 O6

LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

4 REFERENCES IN FILE CA (1907 TO DATE)
 4 REFERENCES IN FILE CAPLUS (1907 TO DATE)
 2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> exp dihydropristimerin/cn

E1	1	DIHYDROPREHELMINTHOSPOROL/CN
E2	1	DIHYDROPRETAZETTINE/CN
E3	0 -->	DIHYDROPRISTIMERIN/CN
E4	1	DIHYDROPRIVEROGENIN A/CN
E5	1	DIHYDROPRIVEROGENIN A 16-ACETATE/CN
E6	1	DIHYDROPRIVEROGENIN A 3,16,22-TRIACETATE/CN
E7	1	DIHYDROPRIVEROGENIN A 3,16,28-TRIACETATE/CN
E8	1	DIHYDROPRIVEROGENIN A 3,16-DIACETATE/CN
E9	1	DIHYDROPRIVEROGENIN A 3,22,28-TRIACETATE/CN
E10	1	DIHYDROPRIVEROGENIN A TETRAACETATE/CN
E11	1	DIHYDROPROGESTERONE-B-CYCLODEXTRIN CLATHRATE/CN
E12	1	DIHYDROPROGESTERONE-ESTRADIOL-17-ENANTHATE MIXT./CN

=> sel l2

E1 THROUGH E3 ASSIGNED

=> index bioscience patents

FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
 FILE 'ENCOMPPAT2' ACCESS NOT AUTHORIZED
 COST IN U.S. DOLLARS

	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	13.07	13.28

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 16:54:48 ON 07 JUL 2006

92 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
 search error messages that display as 0* with SET DETAIL OFF.

=> s E1-E3

4 FILE CAPLUS
27 FILES SEARCHED...
3 FILE TOXCENTER
1 FILE USPATFULL
63 FILES SEARCHED...
2 FILE CAOLD
73 FILES SEARCHED...
76 FILES SEARCHED...
85 FILES SEARCHED...

4 FILES HAVE ONE OR MORE ANSWERS, 92 FILES SEARCHED IN STNINDEX

L3 QUE ("DIHYDROCELASTROL DIACETATE"/BI OR 1262-14-2/BI OR 3022-93-3/BI)

=> file caplus uspatfull

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
1.83	15.11

FULL ESTIMATED COST

FILE 'CAPLUS' ENTERED AT 16:56:49 ON 07 JUL 2006
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=> s E1-E3

L4 5 ("DIHYDROCELASTROL DIACETATE"/BI OR 1262-14-2/BI OR 3022-93-3/BI
)

=> dup rem L4

PROCESSING COMPLETED FOR L4

L5 4 DUP REM L4 (1 DUPLICATE REMOVED)

=> d L5 1-4 ti abs bib

L5 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1
TI Derivatives of pentacyclic nortriterpene quinone methides as compounds
useful in the treatment of inflammatory, neurodegenerative, and neoplastic
diseases
AB The uses of celastrol and pristimerin derivs. in the treatment of
inflammatory, neurodegenerative and neoplastic diseases are disclosed,
including dihydro derivs. of celastrol and pristimerin, such as
dihydrocelastrol and dihydropristimerin and their diacetates.
AN 2004:934338 CAPLUS
DN 141:388762
TI Derivatives of pentacyclic nortriterpene quinone methides as compounds
useful in the treatment of inflammatory, neurodegenerative, and neoplastic
diseases
IN Devlin, J. P.
PA USA
SO U.S. Pat. Appl. Publ., 4 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	----	-----	-----	-----
PI US 2004220267	A1	20041104	US 2004-773903	20040206
PRAI US 2003-445717P	P	20030207		
OS MARPAT 141:388762				

L5 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
TI Celastrols as Inducers of the Heat Shock Response and Cytoprotection

AB Alterations in protein folding and the regulation of conformational states have become increasingly important to the functionality of key mols. in signaling, cell growth, and cell death. Mol. chaperones, because of their properties in protein quality control, afford conformational flexibility to proteins and serve to integrate stress-signaling events that influence aging and a range of diseases including cancer, cystic fibrosis, amyloidoses, and neurodegenerative diseases. We describe here characteristics of celastrol, a quinone methide triterpene and an active component from Chinese herbal medicine identified in a screen of bioactive small mols. that activates the human heat shock response. From a structure/function examination, the celastrol structure is remarkably specific and activates heat shock transcription factor 1 (HSF1) with kinetics similar to those of heat stress, as determined by the induction of HSF1 DNA binding, hyperphosphorylation of HSF1, and expression of chaperone genes. Celastrol can activate heat shock gene transcription synergistically with other stresses and exhibits cytoprotection against subsequent exposures to other forms of lethal cell stress. These results suggest that celastrols exhibit promise as a new class of pharmacol. active regulators of the heat shock response.

AN 2004:1131225 CAPLUS

DN 142:211411

TI Celastrols as Inducers of the Heat Shock Response and Cytoprotection

AU Westerheide, Sandy D.; Bosman, Joshua D.; Mbadugha, Bessie N. A.; Kawahara, Tiara L. A.; Matsumoto, Gen; Kim, Soojin; Gu, Wenxin; Devlin, John P.; Silverman, Richard B.; Morimoto, Richard I.

CS Department of Biochemistry, Molecular Biology and Cell Biology, Rice Institute for Biomedical Research, Northwestern University, Evanston, IL, 60208, USA

SO Journal of Biological Chemistry (2004), 279(53), 56053-56060
CODEN: JBCHA3; ISSN: 0021-9258

PB American Society for Biochemistry and Molecular Biology

DT Journal

LA English

RE.CNT 60 THERE ARE 60 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN

TI Triterpenoid inhibitors of interleukin-1 secretion and tumor-promotion from *Tripterygium wilfordii* var. *regelii*

AB Three new triterpenoids, 2,3,22 β -trihydroxy-21-oxo-24,29-nor-D:A-friedooleana-1,3,5(10)-triene, 2 α ,6 β -dihydroxy-3-oxo-24-nor-D:A-friedooleana-4-ene-29-oic acid and 2,3,7-trihydroxy-6-oxo-24-nor-D:A-friedooleana-1,3,5(10),7-tetraene-29-oic acid, named rheol A, B and C, and nine known triterpenoids were isolated from *T. wilfordii* var. *regelii*. Their structures were established on the basis of the chemical reactions and spectroscopic evidence. Isolated compds. and derivs. were observed to inhibit Epstein-Barr virus early antigen activation and showed potent inhibitory activities against interleukin-1 α and β release from human peripheral mononuclear cells.

AN 1997:423692 CAPLUS

DN 127:173813

TI Triterpenoid inhibitors of interleukin-1 secretion and tumor-promotion from *Tripterygium wilfordii* var. *regelii*

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RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD
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L5 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN

TI Stereochemistry. V. Brominated derivatives of 8-lanostene

AB cf. CA 62, Number 2. A solution of 385 mg. Br in 25 ml. HOAc added to a solution

of 1 g. lanostenone in 50 ml. HOAc containing a few drops of HBr, 100 ml. HOAc added after decolorization, and the solution kept 24 hrs. in the dark gave 100 mg. 2 β -bromo-8-lanosten-3-one (I), m. 170° (Me₂CO), [α]D 159° (all in dioxane), and 600 mg. 2 α -bromo-8-lanosten-3-one (II), m. 139°. A solution of 355 mg. Br in 25 ml. HOAc added to a solution of 1 g. 3 acetoxo-2,8-lanostadiene and 0.2 g. NaOAc in 100 ml. HOAc and the mixture after 3 hrs. poured over ice gave 900 mg. II, [α]D 16°. A solution of 200 mg. 2 α -bromo-8-lanosten-3 β -ol (III) and 100 mg. NaOAc in 25 ml. HOAc stirred 1.5 hrs. with a solution of 400 mg. Na₂Cr₂O₇·2H₂O in 25 ml. HOAc gave 155 mg. II. A solution of 5 g. NaBH₄ in 100 ml. EtOH added to a solution of 2 g. II and 5 g. H₃BO₃ in 150 ml. EtOH and the mixture stirred 3 hrs. gave 1.8 g. III, m. 139°, [α]D 24°. A 10% solution of KOH in EtOH (200 ml.) added to a solution of 1.8 g. III in 200 ml. 2:1 EtOH-C₆H₆ and the mixture stirred 12 hrs. in the cold gave 1.45 g. 2,3 β epoxy-8-lanostene (IV), m. 138-9°, [α]D 113°. A solution of 1 g. IV and 500 mg. LiAlH₄ in 100 ml. dry Et₂O refluxed 3 hrs. gave 200 mg. 8-lanosten-2 β -ol (V), m. 93° (Et₂O-EtOH), [α]D 87° (acetate m. 143-4°, [α]D 87°), and some 8-lanosten-3 β -ol, m. 145°. When the crude mixture from the reduction of 1 g. IV was oxidized with 1.5 g. Na₂Cr₂O₇·2H₂O in 200 ml. HOAc, 675 mg. 8-lanosten-3-one, m. 119-20°, [α]D 68°, and 205 mg. 8-lanosten 2 one (VI), m. 106-7°, [α]D 88°, were obtained. Oxidation of 100 mg. V in HOAc with Na₂Cr₂O₇ gave 85 mg. VI. A solution of 200 mg. VI in 50 ml. boiling EtOH treated with 5 g. Na gave 30 mg. V and 150 mg. 8-lanosten-2 α -ol (VII), m. 104-6° (Et₂O-MeOH), [α]D 50°; m. 100° (Et₂O-MeOH), [α]D 27°. VI (200 mg.) in EtOH stirred 5 hrs. with 100 mg. NaBH₄ gave 170 mg. V and 20 mg. VII. IV (1 g.) in 25 ml. CHCl₃ shaken 15 min. with 20 ml. 48% HBr gave 750 mg. III and 200 mg. 3 α -bromo-8-lanosten-2 β -ol (VIII), m. 77-9° and 103-4° (Me₂CO), [α]D 114°; acetate m. 93° (Et₂O-EtOH), [α]D 90°. Hydrogenation of 100 mg. VIII in EtOAc under 100 atmospheric with Pd-C gave 65 mg. V. VIII (300 mg.) with Na₂Cr₂O₇ and NaOAc in HOAc gave 220 mg. 3 α -bromo-8-lanosten-2-one (IX), m. 140-1° (EtOH), [α]D 146°. IX (200 mg.) shaken with Zn and HOAc 24 hrs. in the cold gave 170 mg. VI. A solution of 200 mg. IX in 20 ml. HOAc treated with 2 drops 48% HBr and the mixture kept 4 hrs. in the dark gave 100 mg. IX and 60 mg. 3 β -bromo-8-lanosten-2-one (X), m. 166-7° (Me₂CO), [α]D 68°. A solution of 200 mg. X in 20 ml. HOAc shaken with Zn 24 hrs. in the cold gave 160 mg. VI. A solution of 200 mg. X and 1 g. H₃BO₃ in 150 ml. EtOH shaken 3 hrs. with a solution of 1 g. NaBH₄ in 50 ml. EtOH gave 180 mg. 3 β -bromo-8-lanosten-2 β -ol (XI), m. 112° (EtOH), [α]D 77°. XI with AcCl in C₆H₅NMe₂ after 3 days in the cold gave the acetate, m. 128-30° (Et₂O-MeOH). A solution of XI in HOAc treated with NaOAc and Na₂Cr₂O₇ gave X. XI treated with 5% alc. KOH gave VI after 3 hrs. in the cold. The structures of many of the compds. were confirmed by uv, ir, N.M.R., and circular dichroism studies. The position of equilibrium between I and II was determined by circular

dichroism

studies to be at 22 \pm 5% I; the equilibrium mixture of IX and X contained 38% IX. The data obtained are sometimes not in complete agreement with those of Barton, et al. (CA 51, 17975e).

AN 1965:9251 CAPLUS

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TI Stereochemistry. V. Brominated derivatives of 8-lanostene

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EXP DIHYDROPRISTIMERIN/CN
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